J. Raptor Res. 31(4):327–332

SIXTEEN YEARS OF LEAD POISONING IN EAGLES, 1980–95: AN EPIZOOTIOLOGIC VIEW

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ABSTRACT.—A 16-yr (1980–95) retrospective study was conducted to assess differences in the prevalence of lead poisoning in Bald (*Haliaeetus leucocephalus*) and Golden (*Aquila chrysaetos*) Eagles admitted to The Raptor Center at the University of Minnesota. These years encompass the period before and after federal legislation was enacted restricting the use of lead shot for hunting waterfowl on federal lands (1991). Of 654 eagle admissions reviewed, 138 cases of lead-poisoned eagles were evaluated for the following: recovery location, blood lead concentration, month of admission, radiographic evidence of lead in the ventriculus and primary cause of admission. The prevalence of lead poisoning in eagles did not change after 1991, but mean blood concentrations of lead in the same population decreased. These findings call into question current theories regarding the sources of lead for eagles and the actual mechanisms by which eagles are poisoned. Lead poisoning is a continuing problem both regionally and internationally, and many variables related to this toxicity have yet to be conclusively defined.

KEY WORDS: *Bald Eagle*, Haliaeetus leucocephalus; *Golden Eagle*, Aquila chrysaetos; *federal legislation*; *lead toxicity*; *upper Midwest*.

Dieciseis años de envenenamiento de cobre en aguilas, 1980–95: un opinión Epizootiologico

RESUMEN.—Un estudio en retrospectivo de 16 años fue conducido para valorar diferencias en la frecuencia de envenenamiento de cobre en *Haliaeetus leucocephalus* y *Aquila chrysaetos* admitidos al Centro de Rapace en la Universidad de Minnesota. Estos años abarcan el tiempo antes y despues de legislación federal que fue promulgada limitando el uso de tiros de cobre para cazar aves de agua en terenos federales (1991). De los 654 aguilas investigados, 138 casos de aguilas envenenados por cobre fueron evaluados por lo siguiente: lugar de recuperación, concentración de cobre en la sangre, mes de admitir, prueba radiographica de cobre en el vebtriculo, la primer razon de admitir. La frecuencia de envenenamiento de cobre en aguilas aumento despues de 1991, pero el promedio concentraciónes de sangre con cobre en la misma población reducieron. Estos descubrimientos llaman la atención de teorias corrientes con respecto la procedencia de cobre para agulas y los mecanismos actuales en la manera que los aguilas estan envenenados. Envenenamiento de cobre es un problema que continua en el región y internacional, y muchos variables relacionados con este tóxico que no esta caracterizado conclusivamente.

[Traducción de Raúl De La Garza, Jr.]

Lead poisoning in raptors has been well documented since the early 1970s (Benson et al. 1974, Jacobson et al. 1977, Redig 1979, Redig et al. 1980). Depending on its severity, lead poisoning causes specific clinical signs such as depression, foul-smelling breath, lime-green feces, nonregenerative anemia, vomiting, diarrhea, ataxia, blindness and epileptiform seizures (Gilsleider and Oehme 1982). Subclinical lead poisoning is not as easily detected, but it may weaken raptors and leave them unable to hunt or more susceptible to mortality from vehicles, power lines, steel traps, etc. (Redig 1979).

Secondary toxicity from consumption of lead-poisoned or contaminated waterfowl is thought to be the predominant source of lead exposure for wintering Bald (*Haliaeetus leucocephalus*) and Golden (*Aquila chrysaetos*) Eagles (Feierabend and Myers 1984, Nelson et al. 1989, Elliott et al. 1992).

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Several studies have shown waterfowl to have a high incidence of lead poisoning (Pattee and Hennes 1983, Anderson et al. 1987, Elliott et al. 1992). Annual losses attributed to lead poisoning have been previously estimated to be 2–3% of the fall population of North American waterfowl (Bellrose 1959). Dabbling waterfowl ingest lead shotgun pellets found in sediments of lakes and marshes. The lead remains in the ventriculus where it is eventually eroded and absorbed into the blood and stored in body tissues. These birds, as well as crippled waterfowl that carry pellets in their muscles from nonlethal firearm injuries, pose a threat of secondary lead toxicity to eagles and other animals that ingest them.

Waterfowl are a significant food source for wintering Bald Eagles (Griffin et al. 1982). Therefore, there is the potential for lead carried in waterfowl tissues either as bound residues or whole shot to be ingested by wintering Bald Eagles and to be a major cause of morbidity and mortality (Feierabend and Myers 1984). The National Wildlife Federation adopted this premise to petition regulatory restrictions on the use of lead shot for hunting waterfowl. In 1991, the use of lead shotgun pellets on all federal waterfowl hunting areas was banned. Between 1986–91, many states including Minnesota and Wisconsin adopted similar statewide restrictions on waterfowl hunting. Since the implementation of the lead ban, the percentage of lead-poisoned eagles admitted to The Raptor Center (TRC) has remained steady or increased. We undertook a 16-yr retrospective study of Bald and Golden Eagles admitted with clinical or subclinical lead poisoning to assess any significant differences in those eagles admitted from 1980–90 (prefederal legislation) and from 1991–95 (postfederal legislation).

METHODS

Screening injured eagles for lead residues is a routine part of the admission protocol at TRC. Whole blood was collected in heparinized syringes and submitted to the College of Veterinary Medicine Diagnostic Laboratory, University of Minnesota, for lead testing. Of 654 Bald and Golden Eagles admitted to TRC from 1980–95, 138 cases of lead poisoning were encountered, and data on recovery location, blood lead concentration, month of admission, radiographic evidence of lead shot in the ventriculus and whether lead toxicity was the primary cause of admission were compiled for each case.

Blood lead concentrations were analyzed by two different methods. The first method, used from 1980–82, was a semiquantitative colorimetric method adapted from the assessment of lead values in bovine blood and liver samples (Hammond et al. 1956). In 1983, we began using a graphite furnace atomic absorption spectrophotometry method (Varian, Model 300+ atomic absorption spectrophotometer, Varian Inc., Australia). Lower limits of detection using the spectrophotometric method were <0.2 ppm. Based on clinical experiences, eagles with blood lead concentrations between 0.2–0.6 ppm were classified as subclinical lead exposure and birds with concentrations between 0.61–1.2 ppm were classified as clinical (treatable) lead poisoning. Concentrations >1.2 ppm were invariably associated with mortality. Blood lead values >1.0 ppm represented acute lead exposure, while blood lead values <1.0 ppm (generally 0.2–0.8 ppm) indicated chronic exposure (Pattee et al. 1981, Pattee and Hennes 1983).

Prevalence of lead exposure in eagles was determined for each year studied and the mean blood concentrations were calculated for Group 1 (prefederal lead shot legislation) and for Group 2 (postfederal lead shot legislation). Significance testing was based on a two-sample *t*-test of unequal variances (Campbell and Machin 1993), while significance testing of blood lead concentration data was based on a chi-squared test for independence (Siegel 1990).

RESULTS

Lead-exposed eagles were recovered from 11 states. Minnesota and Wisconsin submitted the most eagles and the most lead-poisoned eagles. Minnesota submitted 25% and 42%, and Wisconsin 42% and 32% of Groups 1 and 2, respectively (Fig. 1). We tested blood from 654 eagles for the presence of lead during 1980–95. The prevalence of lead exposure and/or lead poisoning for individual years ranged from 3–44%. The mean prevalence for Group 1 was 17.5% versus 26.8% for Group 2 (Fig. 2). The prevalence of lead exposed and/or poisoned Bald and Golden Eagles increased 9.3% between study Groups 1 and 2 but the difference between the two groups was not statistically significant ($P = 0.08$). Only one eagle out of all those admitted had radiographic evidence of lead shotgun pellets in its ventriculus.

Blood lead values ranged from 0.2–8.44 ppm. There was a shift from a higher percentage of acutely poisoned eagles (usually fatal) from 1980–90 (Group 1 = 35% vs. Group 2 = 24%) to a higher percentage of chronically exposed eagles (usually subclinical) from 1991–95 (Group 1 = 50% vs. Group 2 = 64%) (Fig. 3). Here, these differences between the two groups were statistically significant ($P = 0.007$).

Lead exposed eagles were admitted in all 12 mo but more were admitted in November and December with >50% of Group 1 and >40% of Group 2 eagles admitted during these mo (Fig. 4). The pri-

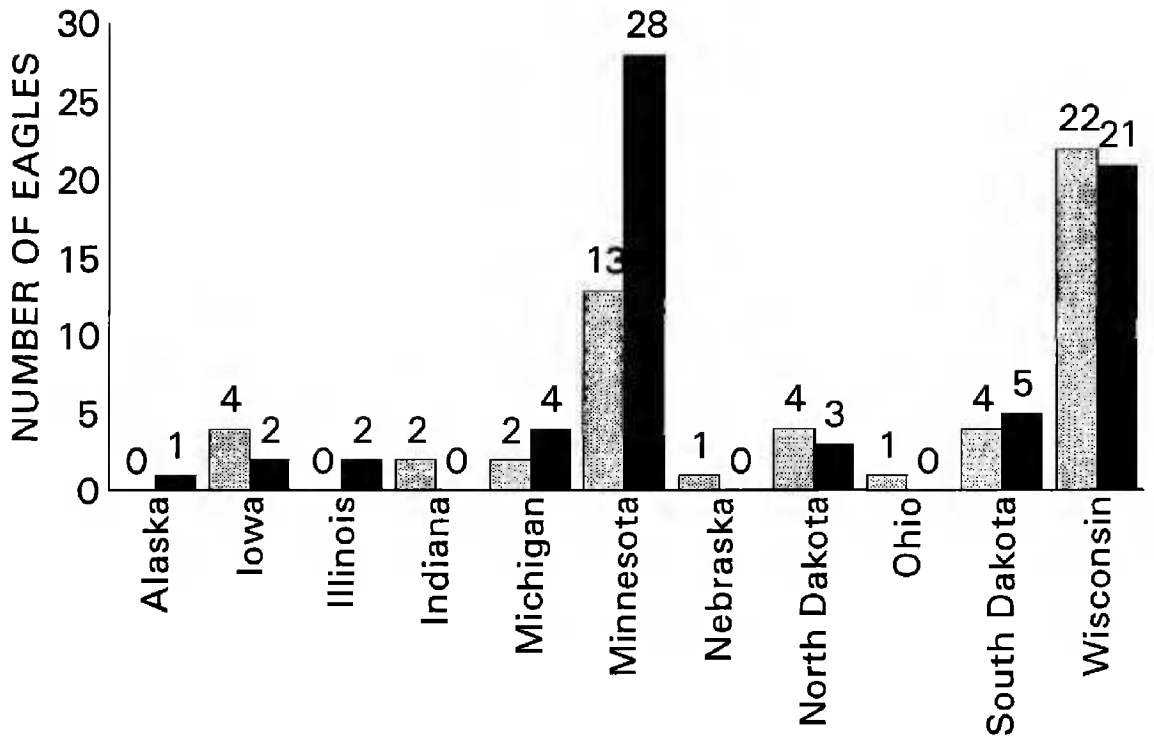

Figure 1. Numbers of eagles admitted to The Raptor Center, University of Minnesota, from each state from 1980–95. Group 1 (prelead shot restriction, $N = 53$) eagles are indicated by shaded bars and Group 2 (postlead shot restrictions, $N = 66$) are indicated by the solid black bars.

mary cause for admission in Group 1 ($N = 72$, but only 55 causes of admission were recorded) was lead toxicity (27%). Secondary and tertiary causes were miscellaneous trauma (22%) and projectile injuries (15%). Miscellaneous trauma (32%) was the primary cause of admission in Group 2 ($N = 66$) with secondary and tertiary causes being projectile injuries (23%) and lead toxicity (17%).

DISCUSSION

Minnesota and Wisconsin enacted a statewide ban on the use or possession of lead shot for waterfowl hunting in 1987. Since then, eagles feeding on waterfowl in these states should not have been exposed to any additional lead shotgun pellets. Hunter compliance has been shown to be as high as 98.9% for harvested ducks and 96.5% for har-

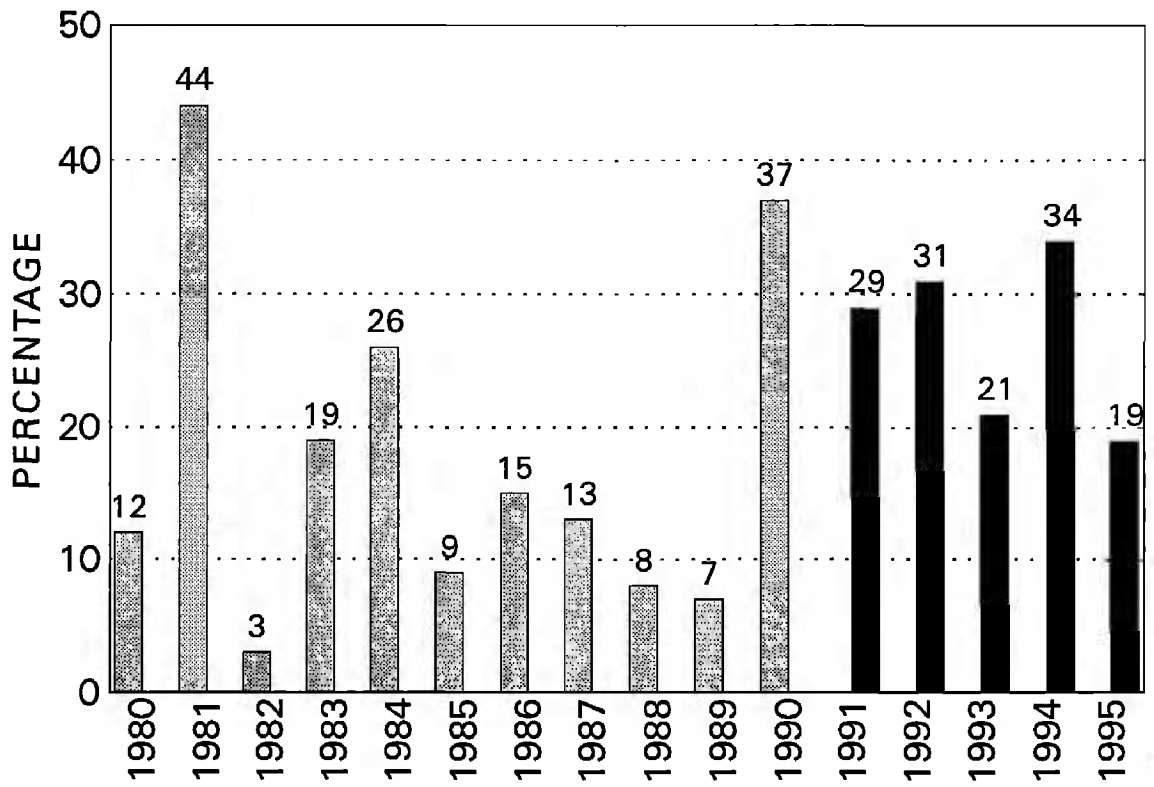

Figure 2. The prevalence of lead poisoning in eagles admitted to The Raptor Center, University of Minnesota, 1980–95. Group 1 (pre1990 lead shot restrictions, $N = 193$, shaded bars) mean 17.5%; Group 2 (postlead shot restrictions, $N = 134$, solid black bars) mean 26.8%.

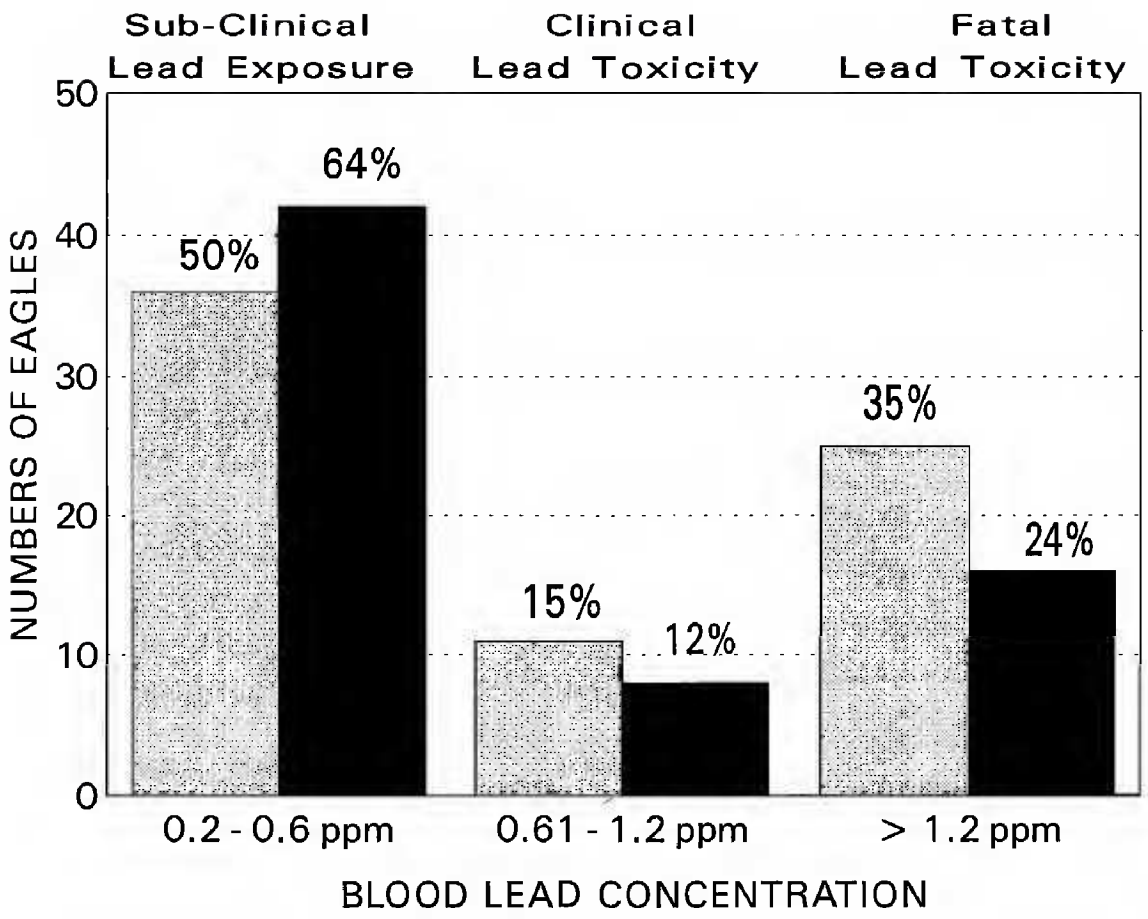

Figure 3. The number and corresponding blood lead concentrations of lead-poisoned eagles admitted to The Raptor Center, University of Minnesota. Group 1 (prelead shot restrictions, $N = 72$) eagles indicated by shaded bars and Group 2 (postlead shot restrictions, $N = 66$) indicated by solid black bars.

vested Canada geese in some areas (Havera et al. 1994). Therefore, in Minnesota and Wisconsin, the number of lead shotgun pellets added to waterfowl habitats after 1987 are presumed to be small. In areas where the use of steel shot has been required, steel has replaced lead in the ventriculi of sampled waterfowl by a ratio of almost 4:1 (DeStefano et al. 1995). One would expect, therefore, the numbers of lead-poisoned eagles would decrease if waterfowl ingestion was the primary source of lead poisoning for wintering eagles.

Our finding that the prevalence of lead in eagles has not changed since the elimination of lead shot indicates that wintering eagles in Minnesota and Wisconsin may rely on other food sources, including a variety of small mammals, birds, fish and carrion (Dunstan and Harper 1975, Platt 1976, Nelson et al. 1989). A variety of small mammals and birds may be hunted year-round and nontoxic shot is not required when hunting these animals.

An additional food source (and potential lead source) may be deer offal containing lead fragments that are left in the field after evisceration by hunters. Deer offal were the source of lead responsible for three of four California Condor (*Gymnogyps californianus*) deaths in the 1980s (Janssen et al. 1986). Highest rates of eagle admissions to TRC were in November and December, a period which coincided with deer seasons in Minnesota and Wisconsin.

Minnesota and Wisconsin are the two states in the upper Midwest region with the largest eagle populations and >50% of all eagles admitted to TRC came from these two states. Eagles admitted from late spring to early fall were probably residents of the local population, but those that were admitted during fall and winter may have been migrants from Canada (Gerrard and Bortolotti 1988). Canada did not restrict use of lead shot for waterfowl hunting during the years included in this study. It is possible that migratory eagles were exposed to lead in Canada, but did not succumb to its effects until arriving on wintering grounds.

Of all eagles admitted to TRC, including those with acute lead poisoning, only one had lead pellets in its ventriculus. Radiographic evidence of the presence or absence of lead in eagles' ventriculi can neither be used to estimate quantitative lead blood concentrations nor be used to demonstrate lead exposure.

Blood lead data supported diagnoses made when eagles were admitted to TRC. Group 1 eagles

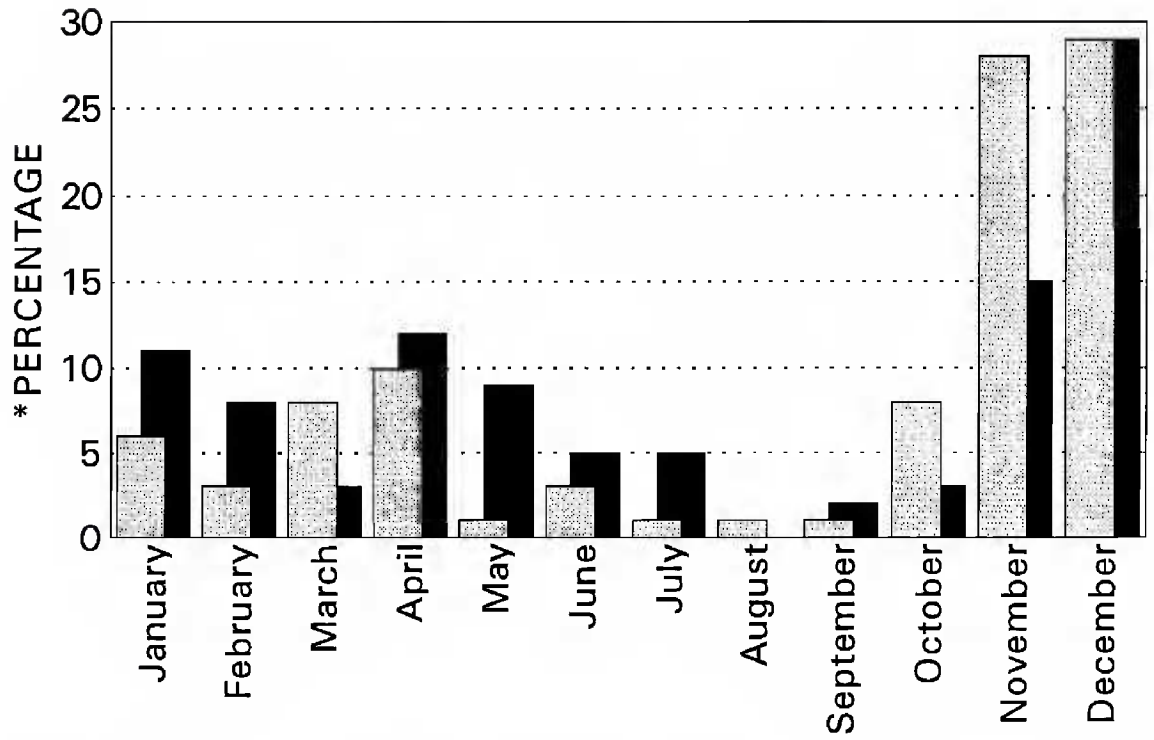

Figure 4. Percentages of lead poisoned eagles admitted to The Raptor Center, University of Minnesota by month. Group 1 (prelead shot restrictions, $N = 72$) indicated by shaded bars and Group 2 (postlead shot restrictions, $N = 66$) indicated by solid black bars.

were more acutely affected and lead poisoning was indicated on admission forms as the primary cause of admission. Blood lead data suggested that Group 2 eagles were chronically exposed, and generally lead poisoning was not cited as the reason for admission. Subclinical or chronic lead exposure usually decreases an eagle's ability to hunt and predisposes it to injury from environmental hazards such as vehicles, power lines and steel traps (Redig 1979) which would explain why the primary reason for admission of these eagles was miscellaneous trauma.

ACKNOWLEDGMENTS

We would like to thank all the staff and volunteers at The Raptor Center for making the data used in this paper available. Also, we would like to thank Tom Arent at the Veterinary Diagnostic Laboratory at the University of Minnesota for conducting laboratory procedures and Dave Kramer for his technical computer skills, patience and support.

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Received 23 January 1997; accepted 13 August 1997